TOXOPLASMA IGG TITRE IN WOMEN WITH BAD OBSTETRIC HISTORY

THESIS

for

MASTER OF SURGERY

(OBSTETRICS & GYNAECOLOGY)

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C E R T I F I C A T E

This is to certify that the work entitled "Tocoplasma Ig G titre in women with bad obstetric history" which is being submitted as a thesis for M.S. (Obstetrics & Gynaecology) by DR. RASHMI GARG has been carried out under my direct supervision and guidance in the Department of Obstetrics and Gynaecology, M.L.B. Medical College, Jhansi.

She has put necessary stay in the Department as required by the regulation of Bundelkhand University, Jhansi.

Dated : 20/11/96

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A C K N O W L E D G E M E N T S

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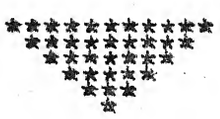
C O N T E N T S



INTRODUCTION

I N T R O D U C T I O N

In the present era of obstetrics and gynaecology more and more attention is being paid to had osbtetric history and to the factor responsible for it.

The term bad obstetric history (BOH) is applied to a pregnant mother where her present obstetric outcome is likely to be affected adversely by the nature of previous obstetric diasaster. The previous fruitless conception should be obstetrically related and as such mishaps to the baby due to some other reason should not come to the purview of BOH.

It include previous history of premature delivery, still births, abortion and congenital malformation. In the developing countries too often the diaster is linked with inadequate or neglected antenatal, intranatal or neonatal care. In obstetrics any complicating factor known or unknown is likely to recur and if it recur in two consecutive pregnancies, the chances of its recurrence in third pregnancy is highly probable when the responsible factor is detected appropriate therapy can be directed so as to prevent repetion of mishaps since women with BOH can experience miscarriage both early and late in pregnancy with a variety of clinical manifestation there is unlikely to be a single aetiology.

Some of important causes of BOH are :-

- Defective germ plasm. Either the spermatozao or ova are defective chromosal abnormalities of embryo are the most common cause of sporadic miscarriage.
- Hormonal deficiency -
  - e.g. Poorly controlled diabeties
  - thyroid dysfunction
  - hypersecretion of luteinizing hormone
  - corpus lupeum deficiency
- Defect in cervicouterine environment
  - cervical incompetence
  - Defective mullerian fusion such as double
  - uterus septate or bibornuate uterus.
  - Uterine fibroid
- Environmental factor
  - heavy alcohol consumption
  - heavy smoking
- Rh isoimmunication
- Chronic maternal illness
  - Chronic hypertension
  - Chronic nephritis
  - Severe anaemia and malnutrition

- Infection - Maternal infection with toxoplasma gondii, cytomegalovirus, rubella, syphilis, ureoplasma urealyticum can cause sporadic pregnancy loss.
- Autoimmune disease
- Idiopathic

Among the various causes of BOH, infections are gaining much importance because the pregnant women and her foetus are susceptible to many infections some of these may be quite serious and life threatening for the mother where as other may have a profound inpact on foetal outcome, and infections are among those few causes of BOH which are preventable and treatable.

Toxoplasmosis is a wide spread infection and has been reported from all geographic area of world. Because most infections in pregnant women are asymptomatic. It seems lagical to screen pregnant women with BOH for toxoplasmosis.

The present study was carried out to find seroepidemiologic aspect of toxoplasmasis in women with previous history of pregnancy loss in form of abortion, preterm, still birth and congenital malformation.

Main aim and objective of study are -

1- To know incidence of toxoplasmasis in pregnant women with bad obstetric history in Bundelkhand area.

2- To know effect of toxoplasma gondii infection on mother, foetus and obstetrical outcome.

3- To offer either pregnancy termination or prenatal therapy in seropositive women who wish to continue their pregnancy.

REVIEW OF LITERATURE

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The term bad obstetric history is applied to a pregnant mother where her present obstetric outcome is likely to be affected adversely by the nature of previous obstetric disaster. The previous fruitless conception should be obstetrically related and as such mishaps to baby due to some other reason should not come to the preview of BOH. It includes previous history of premature delivery, still birth, abortion and congenital malformation.

BOH is a distressing problem affecting approximately 1% of all women. The most significant increase in risk occurs after the first miscarriage. The risk of miscarriage in nulliparous women and those who have had a successful pregnancy rises from approximately 5% to over 20% after one miscarriage. The risk increases there after with each successive miscarriage reaching over 40% after three consecutive losses. (Regan, L.; Breurde, P.R. 1989; Kundsen, U.B., Hansen V., 1991).

Bad Obstetric History can be due to :

There are recognized courses of BOH that include hormonal disorder (thyroid disease and luteal phase defect). Chromosomal anomaly in one or both parents, uterine infection and possibly uterine anatomical defect (Stray Pedersen

and Stray Pedersen 1984; Daya et al 1988; Daly et al 1983; Toth et al 1986).

- Chromosomal abnormality of the embryo is the most common cause of sporadic miscarriage. A women with history of repeated pregnancy loss. Chromosomal abnormalities of embryo occur much less frequently (Bave J., Bave A., 1973).

- Heavy alcohol consumption (Sabal, R.J.; Miller, S.I., 1980; Kline, J.; Shraut, P., 1980).

- Heavy smoking (Parazzinni F., 1987)

- Embryo supposed to implant in avascular part of endometrium in case of uterine septum leading to arrested development (Harger J.H., 1983).

- Defective Mullerian fusion such as double, uterus, septate or Bicornuate uterus (Bennert M.J., 1987).

- Cervical incompetence (DeCherney, A. et al 1987)

- Diethylstillboestrol exposure in utero (Kaufman, R.H. Adam E., 1980).

- Menstrual disorders and infertily are the presenting hallmarks but recent evidence shows that Asherman's syndrome may cause recurrent pregnancy loss (Schenker, J.G et al 1982).

- Myomas are associated with an increased risk for obstetric complication including miscarriages, preterm

labour, ruptured membranes, abnormal fetal presentation (Babaknia A et al 1984).

- A rare endocrine etiology for early pregnancy losses in maternal hyperandrogenicity (Badarau, L. 1972).

- In insulin dependent diabetic women with inadequate glucose control have two to threefold higher rate of recurrent pregnancy loss as compared to general population of women (Whettaker, P.; Tayler, A., 1982).

- Thyroid dysfunction is often an aetiological factor for recurrent pregnancy loss (Winikaff, D., 1982).

- Luteal phase deficiency is an important cause of early pregnancy loss (Csapo, A.I. 1972, 78 & Rabinsersan, D. Taher, M., 1992).

- Mycoplasma Hominis and Ureaplasma urealyticum have been associated with recurrent pregnancy loss (Stey Pedersen, 1984).

- Treponema pallidum is a well known cause of still birth and second trimester abortion (Harter, C.A. 1976). Lyme disease may result in fetal infection and still birth can occur (Weber, K.; Bratzke, H.J. 1988).

- Listeria monocytogenes infection can cause spontaneous abortion, preterm labor and neonatal infection (Rappaport, F.; Rabinovits, M. 1960).

- Herpes simplex virus and cytomegalovirus are two virus which can cause habitual abortion (Kriel, R.L.,1970).

- Malaria during pregnancy is associated with spontaneous abortion, still birth, low birth weight and prematurity (Lewis, R. 1973).

- Primary infection with toxoplasma can cause foetal death and miscarriage (Faulen W., 1990).

- Incompatible ABO group mating may be responsible for early pregnancy wastage and often recurrent but Rh incompatibility is a rare cause of death of foetus before 28th week.

- Chronic hypertension leads to foetal malnutrition preterm labour and abruptio placental which can cause sudden intrauterine death of foetus (Lindheimer, M.D. 1985) and Arias F. 1975, 1979).

- Chronic renal disease can cause placental vascular insufficiency which can cause preterm birth and foetal growth retardation (Cunnirghan, F.C. 1990).

- Anaemia during pregnancy is associated with preterm births, still births and neonatal deaths (Raszkawski, I.; Wajacick, J., 1966).

- Presence of circulating antiphospholipid antibody is a marker for poor pregnancy outcome (Gatenley, P.A. et al 1989).

- Mothers producing antipaternal blocking antibodies that have complement dependent or antibody dependent lymphocytotoxicity. These antibodies have a protective effect and absence of these cause pregnancy rejection (Oksenberg J.R. et al, 1993).

Historical Aspect of Toxoplasma Gondii :

Toxoplasma was discovered by Nicole and Monceaux in 1908 in a small rodent gondii (Clenodactylus gondii) of Africa. Human importance of organism was realised 30 years after i.e. from the year 1939. Although 2 cases were reported in the interval one in 1914. Castellavi from Srilanka and other in 1923 by Jonku. Toxoplasma gondii was first recognized as a cause of congenital infection and disease by Janku in Chachoslavakia in 1923.

We now realize that it is cosmopolitian in the human population and can cause disease. The importance of the organism as a human pathogen has stimulated a huge amount of research in recent years.

Thus the one time obscure protozoan parasite of an obscure African rodent has become one of the most exciting subjects in parasitology.

Toxoplasma gondii is an intracellular protozoan parasite. It was placed in subphylum sporozoa in 1964 by Society of Protozoalogist.

Morphology, Biology and life cycle :

Toxoplasma gondii is a delicate ovoidal, pyriform or crescentic body measuring 4 to 6 microns long by 2 to 3 microns wide with one or both extrimities pointed or rounded. With Giemsa's or Wright's stain it has a blue stained cytoplasm containing a rounded red stained mass of chromatin in the nucleus.

In human infections the organism is found in smear of exudates and in the granulomatous tissue either singly free or intracellular or in cyst like masses, T. gondii is a parasite of endothelial cell, leukocytes, body fluids and tissue cells of the host such as cardiac and skeletal muscle, alveoli, cells of the kidney tubules intestinal mucosa, liver parenchyma : endothelial and reticular cells and neurons.

(Frenkel, J.K., 1973) - Life cycle includes intestinal epithelial (enteroepithelial) and extraintestinal stage in domestic cats and other felines. But extra intestinal stage only in other hosts. Sexual reproduction of toxoplasma occurs while in cat and only asexual reproduction is known while in other hosts.

Extraintestinal stage begins when a cat or other host ingests bradyzoits, ingested tachyzoils or sporocysts also some times are infective. Entero epithelial stage are

initiated when a cat ingests zoitocysts containing bradyzoites, oocysts containing sporozoites or occasionally tachyzoites.

ENTERIC CYCLE :

Hutechison et al (1970) observed schizogenic and Gametogonic stages of T gondii inside epithelial cells of small intestine (ileum) of domestic cat. Large number of oocyte were found in the infected cat faeces.

Schizogony and gametagony occured in the epithelium of tip of intestinal villi. They usually develop in the ileum (the commonest site of infection) but the whole intestine can be affected about 4-29 merozoites are found inside the schizont. The merozoit liberated from the rupture of schizont may continue their cycle of schizogony while other develop into micro and macrogometacytes, after fusion and division these sexual form give rise to oocyte.

EXOENTERIC CYCLE :

The oocyte containing 2 sporocyst are excreted in cat faeces for about 1-2 week. On maturation sporocyst develop into 4 sporozoites resembling trophozoites and become infective to man and other animals. The oocyte after ingestion liberate sporozoite which is heterologous hosts penetrate mucosal cells of the intestine and the prolifera-

tive stage of the parasite and is responsible for causing extensive damage to tissue in which it develops.

According to Hoase, C.A. (1972) two forms of toxoplsma are found in man.

1- Tissue cyst or extracellular form : It may be found free in the tissue fluids. Some of the sporozoits and also the endozoites tend to localise in the central nervous system and the musculature where they are transformed into tissue cysts inside which the parasites also multiplies.

2- Pseudocyst or Intracellular form : It is found in the cells of RE system and many nucleated cells.

MODE OF INFECTION (Transmission):

According to Jacob (1968) human toxoplasmasis can occur in two way.

(A) Congenital : The most tragic form of this disease is congenital toxoplasmasis. Toxoplasma can cross placental barrier from the mother's blood and affect developing foetus.

(B) Acquired :

1) Ingestion of undercooked meat containing tissue cyst. In countries like France where raw meat is popular the infection rate is high (Hughes, H.P.A., 1985).

2) By inoculation (through skin) contact with infected tissues of animals, toxoplasma can penetrate through cracks

and small abrasion in the skin. Sheep and swine may be likely source of infection in man if handling or testing of meat prior to cooking is done - Work, 1971.

3) By accidental ingestion of oocytes that had been shed in a cat's faeces - Flies can contaminate food with viable oocytes for upto 48 hours after contact with cat faeces (Wallace, G.D. 1971).

Clinical feature :

The disease toxoplasmosis has been recognised both as a neonatal infection and as an acquired infection in children and adults. Antibody to toxoplasma is widely prevalent in human through out the world yet clinical toxoplasmosis is less common. Most infection are asymptomatic and mild. Several factors influence this phenomena the virulence of strain of toxoplasma, the susceptibility of the individual host, age of host and degree of acquired immunity of the host.

Symptomatic infection can be classified as acute, subacute and chronic.

In most acute infections the intestine is the first site of infections, first extraintestinal sites to be infected are mesentric lymph node and the parenchyma of liver. Most common symptom of acute toxoplasmosis is painful swollen lymph glands in cervical, supraclavicular and

inguinal regions. This symptom may be associated with fever, headache, muscle pain, anaemia and sometimes lung complications. This syndrome can be mistaken easily for Elue. If immunity develops slowly the condition can be prolonged and is then called subacute, tachyzoites cause extensive lesion in lung, liver, herat, brain and eyes.

Chronic infection results when immunity builds up sufficiently to depress tachyzoite proliferation, cysts can remain intact for years and produce no bbvious, clinical effect, host may develop symptom of chronic encephalitis, blind spots, cyst rupture in retina can lead to blindness. Other are myocarditis with permanent heart damage and pneumonia. In immunocompetent person it can cause disseminated disease.

IMMUNOLOGY OF INFECTION

Quantitative studies of immunoglobin profile reveals that the intitial response in acute acquired toxoplasmosis is the elaboration of Ig M antibodies with in first 20 days of infection, this type of antibody peaks and then diminishes quantitatively with the appearance of specific antibodies of the Ig G type. In chronic toxoplasmosis the antibody is exclusively Ig G (Jones M.H., Sever J.L., 1966).

The fetus in utero responds to infection with T gondii by elaborating Ig M antibodies, This response may result in an overall elevation of Ig M level in the cord serum as well as the presence of specific Ig M antibodies.

DIAGNOSIS :

T. gondii is an obligate intracellular parasite All methods to cultivate it on synthetic media have been unsuccessful. T gondii has been successfully grown in tissue culture of rat embryo heart by Lack (1955) and by other workers in embryonic ovian tissue. Pulvertaft et al 1954 grew the parasite on murine tissue (bone marrow, lymphnode). Intraperitoneal inoculation of a biopsy of lymph node, liver of spleen into mice is useful and accurate.

For many years the routine serological method for toxoplasmosis had been the methylene blue dye test often refered as Babin-Feldman dye test. The disadvantage of this otherwise sensitive test is the necessity of using living organism potentially infectious to the laboratory worker (Sabin & Feldman, 1948).

In direct fluorescent antibody would be the test of choice in the clinical laboratory provided that the equipment for fluorescent microscopy is available. If the laboratory is not so equipped the alternative serological method would be indirect hemagglutination test (Peter G Beach etal 1978).

The complement fraction test can contribute in making a serodiagnostic interpretation from a single serum sample but itself is not a good diagnostic method because of the long delay, upto two months in becoming positive after exposure (Alexand Macdonald 1950).

Demonstration of specific antibody using an enzyme linked immunosorbent assay is easy and accurate method (Van Loon, A.M. et al 1980).

Toxoplasmosis in pregnancy :

Congenital toxoplasmosis was first reported by Jocoby and Sagenin (1948) in Britain. Complement fixing and neutralising antibodies were present in some apparently healthy women (Macdonald 1949).

Cathie & Dudgeon (1949) recognise a number of sign which suggest congenital toxoplasmosis but themselves these signs are insufficient for diagnosis which call for laboratory investigation and to obtain some guidance on the incidence of infection.

Sabin and Feldman (1949) investigated the dye test and complement fixing toxoplasma antibodies in 3 mothers who had given birth to a child with toxoplasmosis and then a normal child. They found that both types of antibodies were transmitted to the normal infant and almost

disappeared after 4 or 5 months, they suggested when a mother has one child with congenital toxoplasmasis her subsequent children are likely to be normal.

Alexander Macdonald (1950) found out of 250 samples of serum from "normal" pregnant women 13 gave positive complement fixation test for toxoplasma antigen in north west England. Out of 12 children who had both chorioretinitis and cerebral calcification 10 gave positive serological tests for toxoplasmosis, they suggested that such symptomless infection must be kept in mind as infection crosses the placenta.

H.G. Farquhar (1950) reported two cases of congenital toxoplasmosis, he described tetralogy of congenital toxoplasmasis, cerebral calcification, hydrocephalaus chorioretinitis and positive serological tests for toxoplasma antibodies he demonstrated antibodies in infected or uninfected children of infected mother.

Jirovee et al (1959) and Langer and Geissler (1960) believe that toxoplasmosis is one of the main infectious cause of repeated abortion in women, other workers believes that women who have congenitally infected child do not have another infected child in subsequent pregnancies, repeated abortion occurs if women developed only an incomplete immunity.

O. Thalhammer (1962) suggested that only when a women is initially infected with toxoplasma during pregnancy can pass the infection to her foetus and the infection can get through the placenta only during the second half of pregnancy, so all women at the end of third month of pregnancy should be tested by toxoplasm in skin test, which is cheap and simple. If foetus is in great danger pyrimethamine 25 mg and triple sulphomonamide 3 gm is given daily, which should be continued for 2 to 3 weeks.

It has long been presumed that material infection with T. gondii had to occur during gestation in order to involve the conceptus. Remington (1963) cultured 34 gravida who exhibited serological evidence of chronic toxoplasmosis and whose pregnancy terminated in abortion, still birth or neonatal death. He recovered the organism in two cases of abortion and in one case of neonatal death. This study demonstrates that a gravida does not have to acquire primary infection during gestation to transmit the organism to the conceptus and persistent parasitemia can occur despite high antitoxplasma antibody level.

Eckerling, Neri and Eylan (1968) studied 40 women with positive serology who previously in 116 pregnancies had produced only 32 surviving infants and after treatment with pyrimethamine and sulphonamide before pregnancy and with

tetracyline and sulphonamide during pregnancy and these 40 women had 42 pregnancies with 41 healthy children and one abortion. In the light of more recent knowledge that tetracyline is potentially teratogenic it should not be used.

Samuel A., Saxan (1971) suggested that subclinical congenital toxoplasmosis may have an adverse effect on intellectual development, so mothers should be screened for toxoplasmosis and treatment with sulfadiazine and pyrimethamine show no evidence of intellectual impairment.

Andrew Czeizel et al (1971) found toxoplasmosis in 7.7 per thousand pregnancy in Hungary. As toxoplasmosis infection occuring in pregnancy effects only 25% of fetuses the rate of fetal disease was 2 per thousand pregnancies and he found no need for routine screening of disease in all pregnant women in Hungary.

Desmonts Georges et al (1974) studied 378 pregnant women with high initial toxoplasma antibody titres or sero conversion during pregnancy, 183 acquired the infection during pregnancy, a rate of 6.3 per 100 pregnancies. There were 11 abortion, 7 infants were still born toxoplasmosis occured in 59 of the non aborted offspring. Severe disease was noted only when maternal infection were acquired during the first two trimesters, later resulted in subclinical or no fetal

infection. Treatment with spiramycin during pregnancy reduced overall frequency of fetal infection but not the overt disease. Mothers with antibodies before they become pregnant had no infected infants.

Babili stray Pedersen et al (1975) carried out a serological screening for toxoplasma antibodies among 10,729 pregnant women in the oslo area and 1007 women in more, frequency of antibody was found to be 12.5% in oslo area and 13.3% in more, frequency was higher in women living in rural area. Incidence of congenital toxoplasmasis was 2% of all pregnancies. No serological difference was found when women with prior histories of either sporadic and habitual abortion were compared to total group of multigravidae among the oslo women.

Raux et al (1976) advised that routine serological screening might be worth while where toxoplasmosis is unduly prevalent e.g. in Paris where 75% of all patients in child bearing age have antibodies or in Brussels, where they are present in 53% of antenatal patients and there are two clinically manifested cases of congenital toxoplasmosis in per 1000 births.

J. Cauvrent et al (1976) studied 14 pair of twin with congenital toxoplasmosis illustrated the importance of

placental lesion in determining the extent of fetal involvement and advised screening for toxoplasmosis in pregnancy.

Peter G Beach et al (1978) screened sera from 95,929 pregnant women for antibodies to toxoplasma gondii with indirect hemagglutination test in oregon, one in every 200 women contracts toxoplasmosis during her pregnancy.

Christopher B Wilson et al (1979) found that most infant born with congenital toxoplasmosis infection are asymptomatic in new born period but develop neurological, intellectual and auditory deficit later in life, so screening of all pregnant women should be done for toxoplasmosis antibodies.

Wilson et al (1980) found that infection late in pregnancy is usually subclinical at birth but of these children most develop convulsion and other neurological sequelae later in life so all pregnant women should be screened for toxoplasmosis and treatment should be given to positive cases.

Kovar and Harvey et al (1981) found that infection during early pregnancy is most damaging although abortion is not common but when infection occurs during the first trimester one fifth of babies will show full syndrome of chorioretinitis, hydrocephaly and patchy crebral calcification, associated with impaired vision, convulsion and severe mental handicap. Since neonatal treatment improves the

immediate prognosis in actue disease but does not reduce subsequent ill health and brain damage, the efficacy of antenatal prophylaxis in this way must await long term follow up studies.

Faulen et al (1984) adviced repeated testing of susceptible mother for toxoplasma antibodies. It would detectseroconversion due to primary infection or initially high titre would indicate that toxoplasma infection may have occured early in pregnancy.

Beattie et al (1984) suggested that in Britain, where meat is usually well or over cooked prevalence rate of toxoplasmosis are lower and routine serology would not be cost effective, e.g. in the west of Scotlant only 25% of population have antibodies and the seroconversion rate in pregnancy is only 0.2% (Williams et al 1981) compared with 0.4% and 0.67% respectively in French and Belgium studies and the incidence of congenital toxoplasmosis is usually 0.06 per 1000 birth.

Faulen et al (1984) suggested that antenatal drug treatment has some value in preventing transplacental infection when it is known that primary maternal infection has occured especially in most dangerous early months of pretnancy, he used spiramycin cyclically as a 3 week course with one week interval.

F. Daffas et al (1985) examined 273 pregnant women at risk of infection, incidence of disease is 3% in population. He found that maternal toxoplasmosis in early pregnancy is less opt to be associated with fetal infection than in maternal infection in late pregnancy. If fetal infection occurs however severe disease is more common in those who acquire infection in first half of gestation. This report demonstrates that prenatal diagnosis of fetal infection in pregnancy at risk is useful by identifying maternal antibody. Treatment given was 3 gm spiramycin daily, termination was done in 3 cases inspite of normal ultrasound finding.

J.G. Kappe (1986) did 20 year follow up of patient with congenital toxoplasmosis and found that new lesion continue to develop well after age of 5 years. Screening of women at risk for toxoplasmosis in pregnancy thus seems advisable.

John L Sever (1987) analysed antibody titre for toxoplasmosis in 23,000 pregnant women in Maryland of the 15 pregnancies with raised antibody titre two children had congenital toxoplasmosis and three were still born, doubling in frequency of deafness, 60% increase in microcephaly and 30% increase in low IQ is associated with presence of high maternal antibody to toxoplasma.

Francois Foresties et al (1988) reported a prospective study of 749 documented cases of maternal toxoplasma infection. Infection was diagnosed antenatally in 39 of 42 fetuses, 24 were terminated. 15 mothers were treated with spiramycin. If foetal infection was demonstrated pyrimethamine and either sulfadoxine or sulfadiazine were added to the regimen, only 2 fetuses developed chorioretinitis, remaining were clinically well, so it was concluded that prenatal therapy in women who wish to continue their pregnancies reduces the severity of the manifestation of the disease.

Orellano N et al (1990) studied incidence of toxoplasmosis in pregnant women in various American cities. He found incidence varied from 3-30%.

Roose et al (1993) after screening 2104 women in Germany found 41.6% women were Ig G positive author concluded that screening was both efficacious and cost effective in their propulation.

Portlong, F. et al (1994) estimated high incidence of 0.5 to 1.5% and 30 to 50% of toxoplasmosis in pregnancy. Among the studied 190 women, two third by seroconversion of toxoplasmosis antibody status and one third by rising Ig G titre plus the presence of Ig M, risk of infection was 4%,

17% and 53% respectively in first, second and third trimester, so antenatal screening is cost effective.

Berrebi, A. et al (1994) reported that maternal infection with toxoplasma gondii can cause infection but not in all fetuses. Furthermore appropriate treatment can prevent foetal infection as the parasite does not cross placenta for 4-8 weeks after the onset of maternal infection so termination of pregnancy is no more indicated in cases of maternal toxoplasmosis.

F. Pratlary et al (1995) studied a cohort of 286 antenatal patients for toxoplasma antibodies, 40 were positive, he concluded importance of making diagnosis of toxoplasmosis antenatally in order to limit the number of medical abortion.

Mehta, S. et al (1995) found incidence of toxoplasma seropositivity in the BOH as 24% where as in control group it was 16%. Incidence of abortion was 28%, preterm delivery 20% and incidence of still birth and congenitally malformed fetuses was 25% in patient of BOH. Among the congenital malformations anencephaly, microcephaly, cleft lip and cleft palate were seen.

Soni, I.J.K. et al (1995) found that the frequency of various hazards in Ig M positive cases was abortion 31.8% preterm birth 22.72%, still birth 13.63% congenital anomaly 4.75% and neonatal birth 4.75%. Thus in pregnant women who were tested Ig M positive, the rate of abortion was tripled and rate of preterm delivery was doubled. It is more common in age group of 24-30 year. 3rd para as compared to 1st and 2nd para. Same incidence among vegetarian and non vegetarian.

Nagar, P. et al (1995) concluded that the incidence of toxoplasmosis was found to be higher in women having repeated abortions, premature births and congenital anomalies. The incidence of repeated abortion in the study was 22%, premature birth was 17.8% and congenital toxoplasmosis was 15%. The maximum age group infected was between 23-27 year. Non vegetarian and patients with contact with pet animals had a definite higher incidence of toxoplasmosis.

MATERIAL AND METHOD

MATERIAL AND METHOD

The present study was carried out in the Department of Obstetrics and Gynaecology, M.L.B. Medical College, Jhansi over a period of one year.

Patients were selected from the out patient department and ward of Department of Obstetrics and Gynaecology of M.L.B. Medical College, Jhansi.

Categorization of cases : Cases were studied into two groups:

1- Control Group :

The control group comprised all the cases attending our out door or admitted in ward with normal pregnancy of different trimesters and non pregnant healthy females of reproductive age group. Total number of cases included in this group was twenty five.

2- Study Group :

This group comprised women with previous history of pregnancy less in form of

- Abortions
- Congenital malformations
- Still births
- Preterm delivery

Total number of cases included in this group was ninty.

HISTORY :

A detail history of each patient was taken followed by thorough general, systemic and local examination as follows :

1- Name, age, OPD No. were recorded

2- Occupational status was considered in order to know socioeconomic status of the patient.

3- Education status of each patient was asked

4- Dietery history was asked wheater vegetarian or non vegetarian

5- History of any addiction e.g. smoking alcohol, tabocco was asked

6- History of present illness was elicited.
An enquiry was made about the duration of pregnancy and onset of sign and symptoms in relation to period of amenorrhae if she come in antenatal period.

7- Past history of fever, lump in body, rashes, any eye complaints, cough, jaundice, diabeties, hypertension was enquired.

8- Family history of diabetes and hypertension was inquired

9- Obstetrical history, previous obstetrical history was taken.

- Total number of time patient was conceived
- Total number of full term pregnancy

- Abortions - duration of pregnancy at time of abortion, type of abortion.
- Congenital malformation - discovered by ultrasonography or by receiving product of conception or foetus.
- Premature delivery
- Still birth
- Number of living children and last child birth or abortion.
- Mode of deliveries, sex, weight of babies and condition of babies at birth and at present were noted.

10- Menstrual history - Date of last menstrual period was asked and expected date of delivery was calculated.

11- Drug history - Any treatment taken in past for any medical disease or for pregnancy losses.

EXAMINATION OF THE PATIENTS

1- General Examination - Thorough general examination was done with special attention to pallor, blood pressure, lymphadenopathy, temperature.

2- Systemic Examination - Brief systemic examination of cardiovascular system, respiratory system, central nervous system and of gastrointestinal system was done. This was to exclude any systemic disease.

3- Obstetrical Examination : Thorough obstetrical examination was done as fundal height, lie presentation and fetal heart rate.

4- Per vaginal Examination : It was done whenever necessary as for confirmation of pregnancy in first trimester and when patient complained of pain during pregnancy.

INVESTIGATIONS :

During the first visit the following investigations was done :

1- Haemoglobin percentage estimation was done using Sahili's method.
2- Total elucocyte count, differential elucocyte count and erytherocyte sedimention rate was done to diagnose any infection.
3- ABO and Rh grouping was done because ABO, Rh incompatibility is one of important causes of BOH.
4- VDRL was done in each patient
5- Complete urine examination routine for albumin and sugar and microscopic for pus cell, RBC or any cast.
6- Fasting and post prandial blood sugar examination was done in each patient.
7- Ultrasound examination of lower abdomen wad done in each patient.

- To see for any congenital malfomation of uterus e.g. double uterus, septate uterus etc.
- To know about any uterine disease e.g. fibroid uterus
- To know gestational age of foetus

We can exclude gross congenital malformation of foetus by ultrasonographic examination, for foetal well being.

Collection of the sample : 3 ml of venous blood was taken in a dry vial with autocloved syringe and needle, blood was allowed to clot then tube was related between palm for 2-3 times and clot separated. Then test tube was placed in a incubator in standing position for half an hour. Test tube is then centrifused and serum was seperated, test of Ig G antibody for toxoplasma gondii was done from the seperated serum.

Sample preparation :

Mix patient's serum - 10 ul

Sample dilution buffer - 1 ml

Serum sample may be diluted at the time of use and stored at 2-8°C before testing for a day.

Methods for detection if Ig G antibody for toxoplasmosis

- Indirect fluorescent antibody test (IFAT)
- Passive hemagglutination test (PHA)

- Methylene blue dye test (MBD)
- Complement fixation test (CF)
- Enzyme linked immuno sorbet assay test (ELISA)

Method used in this study was ELISA

Principle : Toxo Ig G is a sandwich enzyme linked immuno-sorbent assay for qualitative determination of Ig G anti-bodies to toxoplasma gondii in serum.

Immunosorbent assay means that antigen in antibody detection tests are attached to a solid phase (Sorbent). Enzymes are linked to antibodies and react with a substrate indicating the immunological reaction through production of colour.

Procedure : Solid phase ELISA test consists of microtitre strips coated with toxoplasma antigen. If the patient's serum has the relevant specific toxoplasma gondii antibodies they bind to toxoplasma antigen an solid phase. After washing the bound antibodies are sandwiched using HRPO labled anti-human Ig G conjugate resulting in formation of toxoantigen human Ig G HRPO labeled antihuman Ig G conjugate complex. The unbound conjugate is removed by washing and the enzyme linked sandwich complex is revealed by chromagenic substrate the intensity of the colour developed is directly proportional to amount of Ig G toxoplasma gondii antibodies in the serum.

After stapping the reaction with stapping solution absorbance is measured at 450 nm using on ELISA reader. Results of patients samples are obtained by calculation/ comparison using the negative and positive controls.

Calculations :

1- Calculate the mean absorbance reading of negative control (MNC).

2- Calculate the mean absorbance reading of cut off control (MCC).

3- Calculate the mean absorbance reading of medium positive control (MMPC).

The test sample with absorbance values greater than or equal to the MCC are considered positive for Ig G anti toxoplasma gondii antibodies. The test samples with absorbance values less than MCC are considered negative.

If an ELISA reader is not available a visual interpretation of result is possible, A specimen can be considered positive if the colour intensity in the sample well is equal to or stronger than the colour intensity in the cut off control wells.

OBSERVATION

OBSERVATION

The present study includes one hundred and fifteen cases. Cases were divided in two groups. Non pregnant and pregnant. Cases as control group and women of bad obstetric history including abortion, preterm delivery, still birth and congenital anomaly as experimental group. Incidence of toxoplasma Ig G titre positivity was studied in different group and compared.

TABEL - Ia : DIFFERENT TYPE OF CASES IN STUDY GROUP

S.No.	Type of pregnancy loss	No.of cases	Percentage
1-	Abortion	36	40.0%
2-	Preterm delivery	24	26.7%
3-	Still birth	13	14.5%
4-	Congenital malformation	17	18.9%
	Total	90	100%

TABLE - Ib : DIFFERENT TYPE OF CASES IN CONTROL GROUP

S.No.	Type of case	No. of cases	Percentage
1-	Non pregnant	7	28%
2-	Pregnant	18	72%
	Total	25	100%

Table - Ia + Ib shows the distribution of cases in control and study groups. Total number of cases in study group was ninty and in control group was twenty five only.

TABLE - IIa : DISTRIBUTION OF CONTROL GROUP CASES ACCORDING TO THE AGE.

Sl. No.	Age group (In years)	Non Pregnant		Pregnant		Total	
		No.	%	No.	%	No.	%
1-	16 - 19	-	-	2	8%	2	8%
2-	20 - 23	2	8%	4	16%	6	24%
3-	24 - 27	2	8%	5	20%	7	28%
4-	28 - 31	1	4%	4	16%	5	20%
5-	32 - 35	1	4%	3	12%	4	16%
6-	36 - 39	1	4%	-	-	1	4%
	Total	7	28%	18	72%	25	100%

TABLE - IIb : DISTRIBUTION OF STUDY GROUP CASES ACCORDING TO THE AGE.

Sl. No.	Age group (In years)	Abortion		Preterm		Still birth		Congenital anomaly		Total	
		No.	%	No.	%	No.	%	No.	%	No.	%
1-	16 - 19	2	2.2	3	3.4	2	2.2	2	2.2	9	10.0
2-	20 - 23	12	13.2	6	6.6	3	3.3	2	2.2	23	25.4
3-	24 - 27	14	15.5	8	8.8	4	4.4	3	3.3	29	32.0
4-	28 - 31	6	6.6	3	3.3	2	2.2	5	5.5	16	17.7
5-	32 - 35	2	2.2	2	2.2	1	1.1	2	2.2	7	7.7
6-	36 - 39	-	-	2	2.2	1	1.1	3	3.3	6	6.6
6											
	Total	36	41.0	24	26.5	13	14.3	17	17.6	90	100

Table - IIa + IIb shows the distribution of cases according to the age group. Maximum number of cases in control group fall in 24-27 years of age group. Maximum cases of abortion and preterm delivery and still birth were in 24 - 27 years age group and of congenital anomaly in 28 - 31 years of age group.

Age distribution of cases

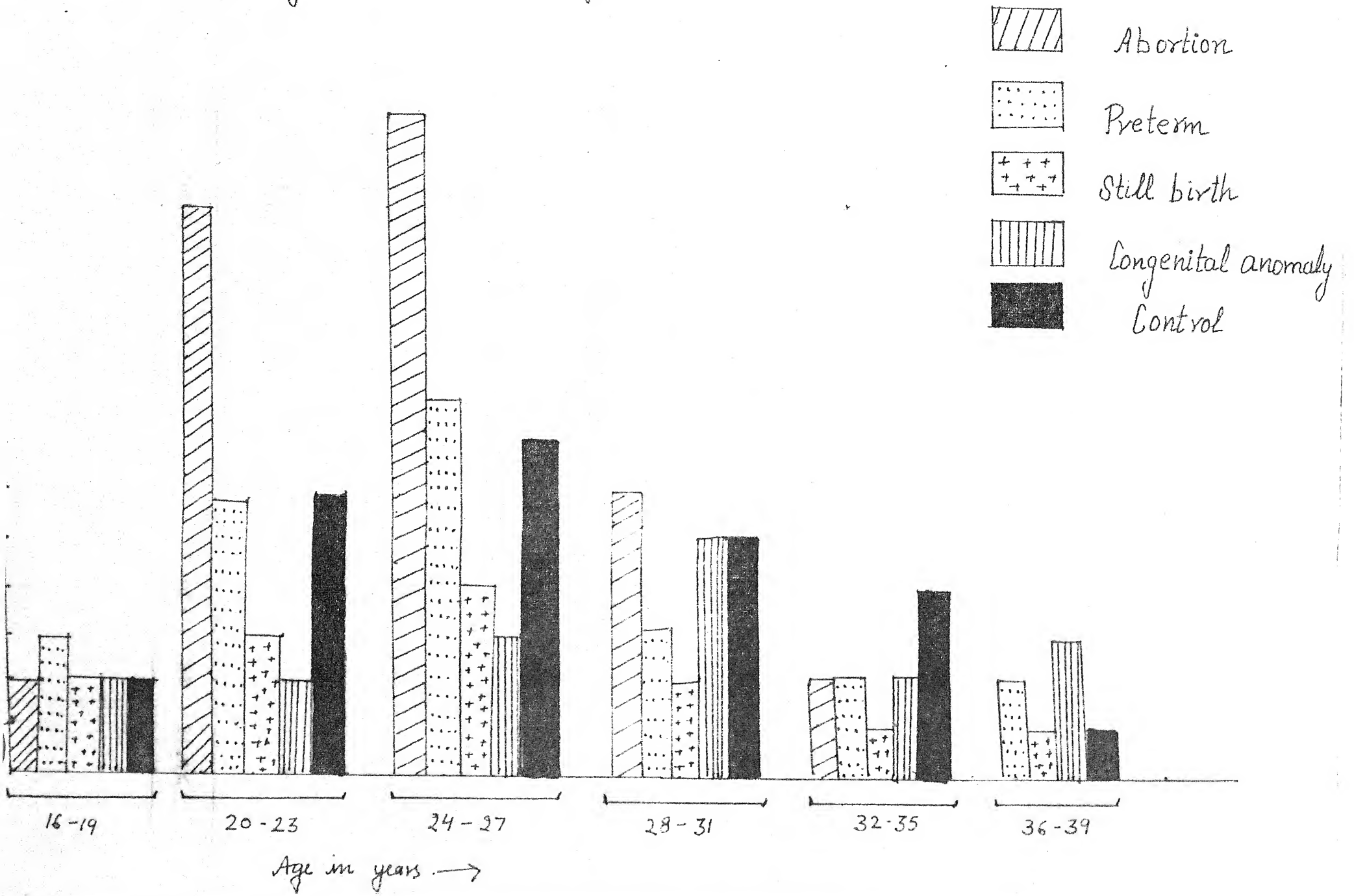

TABLE - III : INCIDENCE OF TOXOPLASMA SEROPOSITIVITY IN STUDY AND CONTROL GROUPS

Sl. No.	Group	No.of cases	Negative		Positive	
			No.	%	No.	%
1-	Study	90	68	75.6%	22	24.4%
2-	Control	25	24	96.0%	1	4.0%
	Total	115	92	80.0%	23	20.0%

Table - III shows that incidence of toxoplasma seropositivity was six times (24.4%) more in study group which includes patients of BOH as compared to women of control where it was 4%.

TABLE - IV : TOXOPLASMA SEROPOSITIVITY IN RELATION TO THE TYPE OF PREGNANCY LOSS.

Sl. No.	Type of Pregnancy loss	No.of cases	Negative		Positive	
			No.	%	No.	%
1-	Abortion	36	24	66.7%	12	33.3%
2-	Preterm delivery	24	19	79.2%	5	20.8%
3-	Still birth	13	10	76.9%	3	23.1%
4-	Congenital malformation	17	15	88.3%	2	11.7%
	Total	90	68	75.6%	22	24.4%

Toxoplasma seropositivity in relation to the type of pregnancy loss

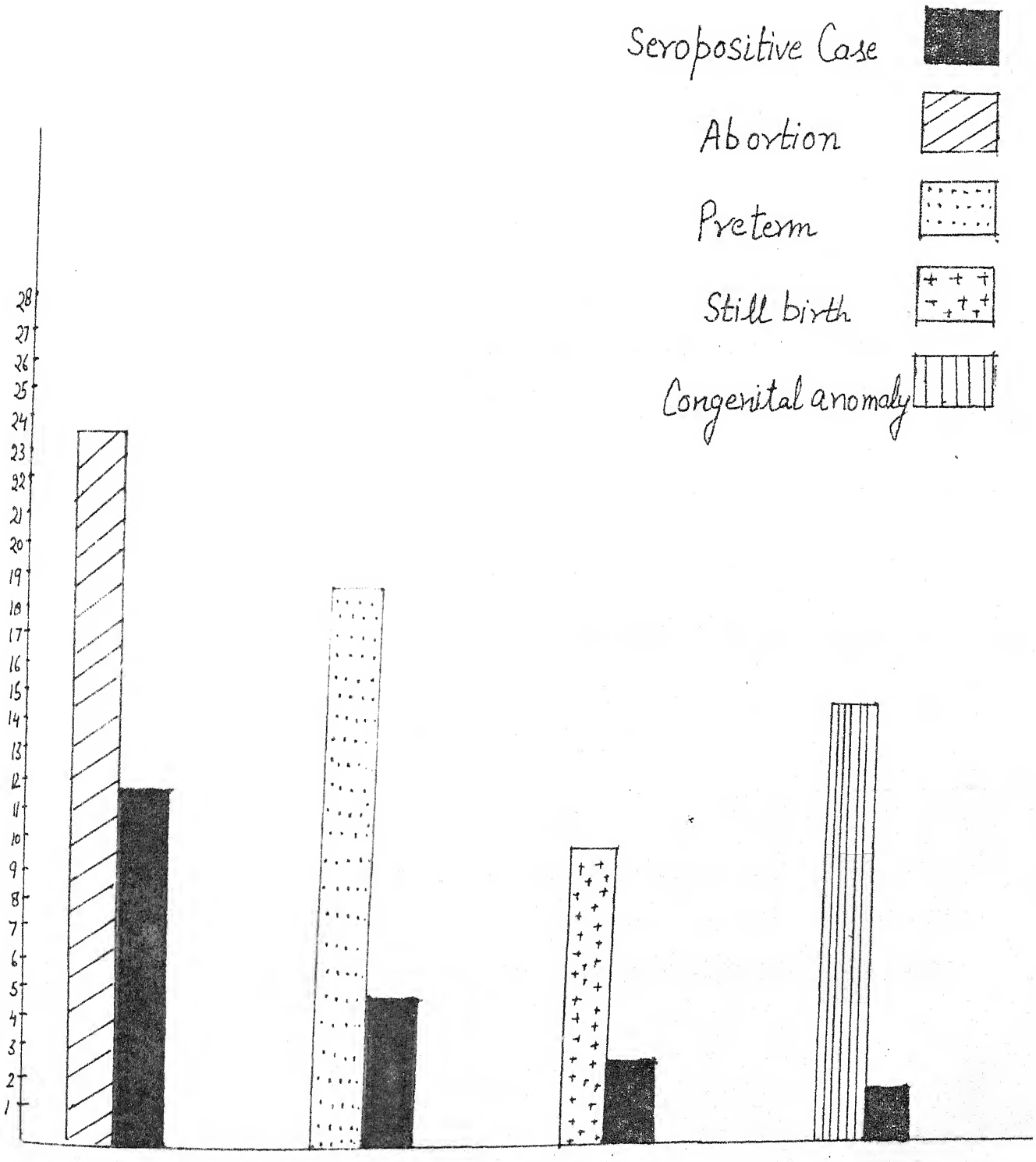

Table - IV shows that maximum seropositivity for toxoplasma was present in abortion cases, (33.3%), second highest in still birth (23.1%) than 20.8% and 11.7% respectively in case of preterm delivery and congenital malformation.

TABLE - V : DISTRIBUTION OF CASES OF STUDY GROUP ACCORDING TO TOXOPLASMA SEROPOSITIVITY AND ITS RELATION TO TYPE OF ABORTION

Sl. No.	Type of abortion	Total No.of cases	Number of positive cases	Percentage
1-	Habitual	21	5	23.8%
2-	Sporadic	15	7	46.6%
	Total	36	12	33.3%

Table - V shows that seropositivity of toxoplasma in relation to the type of abortion. Sporadic abortion was twice as common as compared to habitual abortion in seropositive cases.

TABLE - VI : DISTRIBUTION OF SEROPOSITIVE CASES ACCORDING TO TYPE OF CONGENITAL MALFORMATION.

Sl. No.	Congenital malformation	No.of cases	Positive cases No.	Positive cases %
1-	Anencephaly	9	1	11.1%
2-	Hydrocephaly	3	-	-
3-	Microcephaly	2	-	-
4-	Myelocaele	1	-	-
5-	Cleft lip & palate	2	1	50.0%
	Total	17	2	11.7%

Table - VI shows that among the cases of congenital malformation, anencephaly and cleft lip & palate were having maximum incidence of seropositivity, Hydrocephaly, microcephaly and myelocaele showed no relation to toxoplasma infection.

TABLE - VII : DISTRIBUTION OF SEROPOSITIVITY CASES ACCORDING TO THE AGE.

Sl. No.	Age group (In years)	Control group			Study group		
		Total No.	Positive case No.	%	Total No.	Positive case No.	%
1-	16 - 19	2	-	-	9	1	11.1%
2-	20 - 23	6	1	16.6%	23	5	21.7%
3-	24 - 27	7	-	-	29	11	37.9%
4-	28 - 31	5	-	-	16	3	18.7%
5-	32 - 35	4	-	-	7	1	14.4%
6-	36 - 39	1	-	-	6	1	16.6%
	Total	25	1	4%	90	22	24.4%

Table - VII shows that only one case was positive for toxoplasma in control group in age group between 20 - 23 years, whereas in study group maximum 11 cases (37.9%) were positive in 24 - 27 years age group. 32.8% cases belonged to the age group of less than 23 years and no patient was found above the age of 39 years.

Age distribution of Seropositive and negative cases in Study and Control group

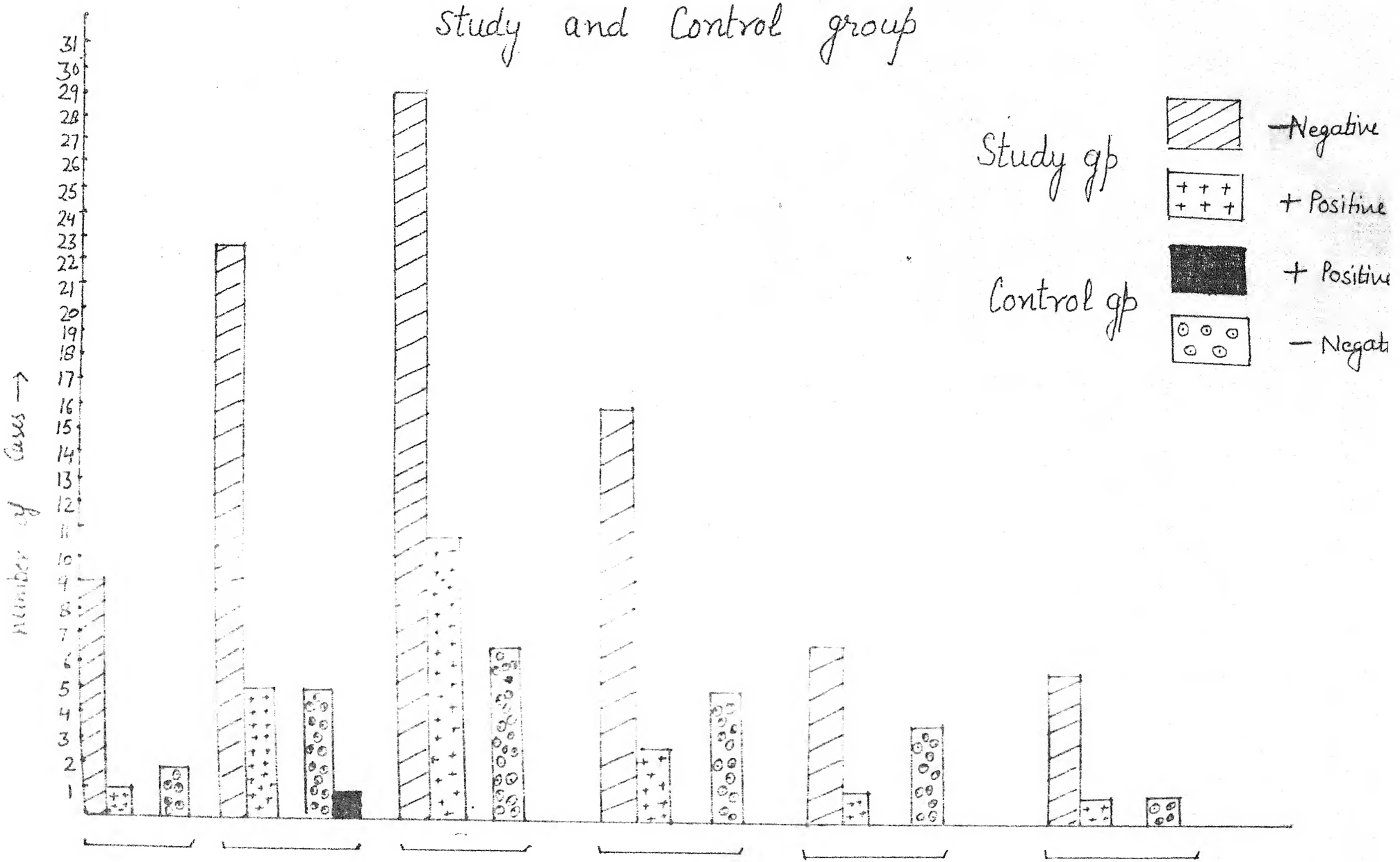

TABLE - VIII : SEROPOSITIVITY IN CASES OF ABORTION ACCORDING TO PARITY

Sl. No.	Gravida	Total No.of cases	Positive cases	
			No.of cases	Percentage
1-	First & Second	8	2	25.0%
2-	Third & Fourth	14	6	42.8%
3-	Fifth & Sixth	6	2	33.3%
4-	Seventh & Eight	4	1	25.0%
5-	Ninth & Tenth	4	1	25.0%
	Total	36	12	33.3%

Table - VIII shows that seropositivity in cases of abortion was maximum (42.8%) in third and fourth gravida; second highest incidence of seropositivity was seen in fifth and sixth gravida (33.3%) where as in first and second gravida it was only 25%.

TABLE - X : RURAL AND URBAN AREA DISTRIBUTION OF SEROPOSITIVE CASES.

Sl. No.	Study Group	Total No.of cases	Rural		Urban	
			No.	%	No.	%
1-	Positive	22	15	70.4%	3	31.8%
2-	Negative	68	43	63.2%	25	36.8%
	Total	90	58	64.5%	32	35.5%

Table - X shows higher incidence of seropositivity in rural area (70.4%) as compared to that in urban area (31.8%).

TABLE - XI : DISTRIBUTION OF SEROPOSITIVE CASES ACCORDING TO DIETARY HABITS

Sl. No.	Study group	Total No.of cases	Vegetarian		Non vegetarian	
			No.	%	No.	%
1-	Negative	68	38	55.8%	30	44.2%
2-	Positive	22	9	40.9%	13	59.1%
	Total	90	47	52.2%	43	47.7%

Table - XI shows that there was higher incidence of seropositivity (59.1%) among non vegetarian as compared to seronegative (44.2%).

TABLE - XII : DISTRIBUTION OF SEROPOSITIVE CASES ACCORDING TO HISTORY OF HAVING PETS

Sl. No.	Study group	Total No.of cases	No pets		Pets	
			No.	%	No.	%
1-	Negative	68	61	89.7%	7	10.2%
2-	Positive	22	19	86.3%	3	13.6%
	Total	90	80	88.9%	10	11.1%

Table - XII shows that there was slightly higher incidence (13.6%) of seropositivity in women having pets.

D I S C U S S I O N

DISCUSSION

Among the various causes of bad obstetric history (BOH) infections are gaining much importance now a days. It if well known that toxoplasmosis can cause pregnancy losses in the form of abortion, preterm delivery, still birth and congenital malformation. The present study was carried out to find out incidence of toxplasmosis in pregnant women with bad obstetric history and to know effect of toxoplasma gondii infection on mother, foetus and obstetrical outcome.

In the present study majority of seropositive cases (37.9%) were in 24-27 years of age range, 32.8% cases belonged to the age group of less than 23 years and no patient was found above the age of 39 years. Soni, I.J.K. et al (1995) reported that seropositivity was more common in age group of 24-30 years. Nagar, P. et al (1995) also suggested that maximum age group infected was between 23-27 years.

Maximum number of seropositive cases were third and fourth gravida. Similar observation was made by Soni, I.J.K. et al (1995).

In our study incidence of toxoplasma seropositivity was 24.4% in study group which was six times more as compared to women of control group where it was 4%. Our findings were similar to the finding of Mehta, S. et al (1995) who found incidence of toxplasma seropositivity 24% in BOH and 16% in control and Soni, I.J.K. et al (1995) who found incidence of toxoplasmosis to be 27.5% in study group and 5% in control group.

Raux et al (1976) reported that 53% of Ante natal patients have antibodies of toxoplasma in Paris. F. Doffas et al (1985) found 3% incidence of disease in pregnant women, Orellano, N. et al (1990) studied incidence of toxoplasmosis in pregnant women in various American cities. He found that incidence varies from 3-30%. Roose et al (1993) in Germany found 41.6% women were Ig G positive, F. Pratlong et al (1995) studied a cohort of 286 with history of abortion for toxoplasma antibodies, 13.9% were positive. Thus it seems logical to do screening for toxoplasmosis in women bad obstetric history.

In present study maximum seropositivity (33.3%) for toxoplasma was present in the cases of abortion, second highest in still birth 23.1% than 20.8% and 11.7% respectively in cases of preterm delivery and congenital malformation.

Our findings are consistent to the study made by previous workers. According to Mehta, S. et al (1995) incidence of abortion was 28%, preterm delivery 20% and incidence of still birth and congenitally malformed foetuses was 25% in patients of BOH. Soni, I.J.K. et al (1995) found that frequency of various hazards in seropositive cases was abortion 31.8%, preterm birth 22.7%, still birth 13.6% and congenital anomaly 4.75%. The incidence of repeated abortions in study made by Nagar, P. et al (1995) was 22%, premature birth was 17.8% and congenital toxoplasmosis was 15%.

Jirovee et al (1959) believe that toxoplasmosis is one of the main infection cause repeated abortion in women. Remington (1963) exhibited serological evidence of chronic toxoplasmosis where pregnancy terminated in abortion, still birth or neonatal death. Desmonts George et al (1974) also found that toxoplasmosis can cause abortion & still birth. John L. Sever (1987) found that in 15 pregnancies with raised antibody titre, two children had congenital toxoplasmosis and three were still born. Our view is also supported by Cech et al (1960) who did skin test for toxoplasmin in 379 women whose pregnancy resulted in various types of pregnancy loss and Hingorani, V. et al (1960) found association of toxoplasma with pregnancy wastage in 14% of their cases.

Alexander Macdnnal et al (1950) found that from 13 pregnant women with toxoplasma antigen, 12 children were effected. They suggested that such symptomless infection must be kept in mind as infection crosses the placenta.

In our study, we found that sporadic abortion was twice as common as compared to habitual abortion in seropositive cases. Our finding of toxoplasma seropositivity in relation to type of abortion are in agreement with the view of Mehta, S. et al (1995) who reported 25% of habitual abortion cases seropositive where as in cases of sporadic abortion seropositivity was 40%

A Kimbell (1971) found association of toxoplasma with spontaneous abortion but not with recurrent abortions. He found that toxoplasma infection is not seen in more than one child of the same mother which indicate that primary infection with toxoplasma has a significant association with pregnancy wastage, chronic infection limits further transmission of disease.

Sabin & Feldman et al (1949) suggested when a mother has one child with congenital toxoplasmosis her subsequent children are likely to be normal.

Langer and Geissler et al (1960) believe that women who have congenitaly infected child to have another

infected child in subsequent pregnancies. Repeated abortions occur if women developed only an incomplete immunity.

In present study, among the cases of congenital malformation, anencephaly and cleft lip palate were having maximum incidence of seropositivity. Hydrocephaly, microcephaly and myelocoel showed no relation to toxoplasma infection, our findings are similar to the findings of Mehta, S. et al (1995) who found Anencephaly and Cleft lip palate in seropositive cases but they also found microcephaly in seropositive cases which was however not seen in our study.

Farquhar, H.G. (1950), Kimbell, A. (1971) and Hanely et al (1981) showed a positive relationship between toxoplasma and hydrocephaløus, which was also not seen in our study.

John L. Sever (1987) found doubling in frequency of deafness, 60% increase in microcephaly and 30% increase in low IQ. It was associated with presence of high maternal antibody to toxoplasma. Microcephaly was not seen in our study.

In our study we found higher incidence of seropositivity in rural area (70.4%) as compared to urban area (31.8%). In the study conducted by Babili Stray Pedersen (1975) of 10,729 pregnant women in Oslo and More area,

higher incidence of seropositivity in rural population was noted. Higher incidence of seropositivity in rural area could be due to lower resistance to infections, unhygeinic living condition and improper processing of meat in that area.

In our study we found higher incidence of seropositivity (59.1%) among non vegetarian as compared to seronegative cases (44.2%). Nagar, P. et al (1995) also found definite higher incidence of toxoplasmosis in non vegetarians, while Soni, I.J.K. et al (1995) reported same incidence among vegetarian and non vegetarian.

Beattie et al (1984) suggested that in Britain where meat is usually well or over cooked prevalence rate of toxoplasmosis is lower as compared to French & Balgium.

Hughes, H.P.A. (1985) also suggested that infestion of undercooked meat containing tissue cyst can cause disease. In countries like France where raw meat is popular the infection rate is high.

Work, 1971 found that contact with infected tissues of animals toxoplasma can penetrate through cracks and small abfasion in the skin, Sheep and swine may be likely source of infection in man, if handling of meat prior to cooking is done.

Frenkel, J.K. (1973) observed that sexual reproduction of toxoplasma occur in cat & other felines. Large number of oocyte were found in infected cat faeces and Wallace, G.D. 1971 found that disease can occur by accidental ingestion of oocyte that had been shed in a cat's faeces. Flies can contaminate food with viable oocytes for upto 48 hours after contact with cat faeces so they found higher incidence of toxoplasmosis in women with contact with pets. In our study we found higher incidence of seropositivity in women having pets. Similar findings were reported by Nagar, P. et al (1995).

SUMMARY AND CONCLUSION

SUMMARY AND CONCLUSION

The present study entitled "Toxoplasma Ig G titre in women with bad obstetrics history" was conducted in the Department of Obstetrics and Gynaecology, M.L. B. Medical College, Jhansi. Cases were selected from the out patient department and ward of Department of Obstetrics and Gynaecology of M.L.B. Medical College & Hospital, Jhansi.

This study included 115 cases aged 16 year to 39 years of age. 90 women having bad obstetric history including abortion, preterm delivery, still birth and congenital anomaly were taken as study group and control group consisted of 25 women. Toxoplasma Ig G titre was measured by ELISA technique.

Following conclusion has been drawn from this work :-

1- Maximum seropositivity (37.9%) was seen in 24-27 years of age group and maximum number of seropositive cases were third and fourth gravida.

2- In our study incidence of toxoplasma seropositivity was 24.4% in study group which was six times more as compared to women of control group where it was 4%.

3- Maximum seropositivity (33.3%) for toxoplasma was present in the cases of abortion, second highest in still birth 23.1% than 20.8% and 11.1% respectively in cases of preterm delivery and congenital malformation.

4- Sporadic abortion was twice as common as compared to habitual abortion in seropositive cases. This could be due to the fact that women developed an incomplete immunity for toxoplasma infection.

5- Among the cases of congenital malformations, anencephaly and cleft lip palate were having maximum incidence of seropositivity, Hydrocephaly, microcephaly and myelocoel showed no relation to toxoplasma infection.

6- Higher incidence of seropositivity was found in rural area (70.4%) this could be due to lower resistance to infections, unhygeinic living condition and improper precessing of meat in that area.

7- Non vegetarian showed higher incidence of seropositivity (59.1%) as compared to vegetarians (40.9%), this could be due to ingestion of under cooked meat

containing tissue cyst or penetration of toxoplasma through cracks and small abrasion in the skin, if handling of meat prior to cooking is done.

8- We found higher incidence of seropositivity in women having pets may be due to accidental ingestion of oocyte that had been shed in cat's faeces.

In conclusion toxoplasmosis is a significant cause of pregnancy wastage, in the form of abortion, preterm, still birth and congenital malformations. Thus is seems logical to do screening for toxoplasmosis in women with bad obstetric history.

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